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The following are abstracts of papers presented at the Annual Scientific Meeting of the Thoracic Society of Australia held in Perth, Western Australia, 2-4 May, 1983.

NEUTROPHIL CHEMOTACTIC ACTIVITY FOLLOWING THE INHALATION OF ULTRASONICALLY NEBULISED WATER

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An increase in serum of a high molecular weight neutrophil chemotactic factor (NCF) has been described in patients with exercise and allergen induced asthma. The increase in NCF after exercise has been shown to be inhibited by sodium cromoglycate suggesting that NCF may reflect mast-cell activation.

We carried out a study in seven asthmatics and five normal subjects to determine if the change in airways resistance following the inhalation of ultrasonically nebulised water (UNH_2O) is also associated with an increase in neutrophil chemotactic activity.

Eight ml of blood was collected from an antecubital fossa vein and allowed to clot on glass for three hours at 4°C. The serum was collected and heated at 56°C for 30 min, then stored at −80°C until analysis. Samples were collected at rest, immediately at the end of challenge, 5, 10, 15, 30 and 60 min. later. Neutrophil chemotactic factor activity was assayed in a modified Boyden chamber in which a micropore filter separates a neutrophil suspension from a 20 percent dilution of test serum. Following incubation the filter is removed and examined by microscopy for evidence of neutrophil migration.

A MistO$_2$gen Ultrasonic Nebuliser (EN143A, California) which delivers approximately 1 ml of water per 10 L of aerosol inhaled was used for the challenge. Forced expiratory volume in one second (FEV_1), forced expiratory flow rate over the middle half of the vital capacity (FEF25-75) and the flow rate at 50% of the vital capacity (V50) were measured using a Cavitron (SC-20 Spirometer, Anaheim, California).

Measurements were made in triplicate at rest and 30 sec. after the inhalation of a known volume of nebulised H_2O. The mean ± SD FEV_1 at rest (expressed as a percentage of the predicted value) was 80.2% ± 21.8 for the asthmatics and 104.3% ± 19 for the normal subjects. Following the water challenge the mean maximum reduction in each measurement was calculated and expressed as a percentage of the pre-challenge value. For the seven asthmatics the mean maximum fall ± 1 SD for FEV_1 was 41.6% ± 11.5, for FEF25-75 46.4% ± 4.2 and for V50 51.0% ± 5.7. The mean ± 1SD delivered dose of H_2O required to induce the maximum recorded change in FEV_1 was 6.8 ml ± 5.5. For the normal subjects the reduction in FEV_1, FEF25-75 and V50 was less than 20% of the pre-challenge level after the inhalation of 33 ml of H_2O.

There was no significant difference in the pre-challenge levels of neutrophils per 10 high power fields (N10HPF) between the asthmatic and normal subjects. In the 30 min after challenge there was a mean ± 1 SD maximum increase in N10HPF of 179 ± 118 in the asthmatics and 42 ± 15.5 in the normal subjects ($p < 0.05$). Sixty minutes after challenge the values for N10HPF had returned to within 8% of the resting levels.

We concluded that the airways obstruction induced by the inhalation of UNH_2O is associated with an increase in serum neutrophil chemotactic activity.

RELATIONSHIP BETWEEN AIRWAY SMOOTH MUSCLE VOLUME AND HISTAMINE REACTIVITY IN VIVO/IN VITRO IN HUMAN AIRWAYS

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Non specific airway hyperreactivity is now recognised as a characteristic feature of human asthma. Alteration in the smooth muscle within the airways has been suggested as the basis of hyperreactivity.

The present study examined lung function and nonspecific bronchial reactivity in 12 patients with chronic obstructive lung disease prior to resection of lung tissue for carcinoma. On the day before surgery and while on no bronchodilator medication, all subjects performed a progressive histamine inhalation test according to the method of Cockcroft *et al.* The inhalation concentration-response curve was terminated when the FEV_1 had fallen by 20% from resting levels or an inhaled concentration of 16 mg/ml had been reached. The PC_{20} was calculated as that concentration of histamine which produced the 20% fall in FEV_1.

Following surgical removal of the lung, portions of segmental or subsegmental airways were dissected free of lung parenchyma and placed in Krebs-Henseleit solution aerated with 95% O_2 and 5% CO_2. The airways segments were then cut into spirals, placed in organ baths maintained at 37°C and attached to isometric transducers under an initial tension of 2 g. Changes in tension resulting from the addition of cumulative concentrations of histamine were recorded on a Beckman polygraph. Responses to histamine were expressed as a percentage of the maximum response and the EC_{50} calculated. Each piece of bronchial tissue

Ovenhandlers also had a greater prevalence of increased bronchial reactivity ($PD_{20} < 30$ μmoles) (56%) than doughmakers (29%) and positive skin tests to wheat (44% vs. 0%, $p < 0.005$). The frequencies for general bakers were intermediate (41% and 17% respectively, NS).

This study demonstrates that bakers have evidence of allergic respiratory disease that is related to their occupational exposure to cereal antigens.

ROLE OF FOOD ADDITIVES (SODIUM METABISULPHITE AND SALICYLATES) IN CHRONIC CHILDHOOD ASTHMA

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We have studied the role of two commonly ingested food additives/chemicals, the preservative sodium metabisulphite (MBS) and aspirin (ASA), in 29 children with moderate-severe childhood asthma. All 29 were challenged, single blind, in the pulmonary function laboratory with MBS (capsule form and solution), ASA and placebo. For one week prior to the challenge, and during the challenge period, all 29 were prescribed to full elimination diet. Following the challenges, positive responders to MBS were placed on a diet which excluded MBS containing foods. ASA positive patients were prescribed a diet excluding natural salicylates and advised to avoid aspirin containing medications. After three months on these restricted diets the children were reassessed to determine any therapeutic response.

Sixty six percent (19/29) had a positive immediate challenge (>20% fall in FEV_1) to metabisulphite and 21% (6/29) had a positive immediate challenge to aspirin. After three months on the restricted diet, 4/19 children on MBS-free diet and 1/6 on salicylate-free diet had objective signs of improvement; namely, a reduction in either steroid or bronchodilator therapy. However, compliance with the diet during these three months was poor, particularly with the aspirin positive children.

We have demonstrated that two commonly ingested chemicals can provoke bronchospasm in asthmatic children. However, elimination of these substances from the diet is difficult and does not, in general improve the child's asthma.

THE EFFECT OF PASSIVE CIGARETTE SMOKING ON ASTHMATIC PATIENTS

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The aim of this study was to examine the effect of passive inhalation of cigarette smoke on airways function in asthmatic patients. Six subjects with bronchial asthma and a history of chest tightness on passive exposure to cigarette smoke were studied. The subjects had well-controlled, mild to moderate asthma. They abstained from beta-2 agonists and inhaled corticosteroids for at least six hours prior to the provocation test, from oral theophylline for 12 hours, from slow release theophylline and sodium cromoglycate for 24 hours and from antihistamines for 48 hours prior to study days. On the first day, baseline FEV_1, FVC, MMEFR and peak flow rate readings were undertaken and the patient then sat in a seven cubic metre room for 60 minutes during which time a mechanical device linked to a rheostat was run but no cigarette smoke was produced. Lung function measurements were repeated at 15, 30, 45 and 60 minutes in the room and thereafter every 15 minutes for two hours. On the second day, the same lung function parameters were measured and the patient spent 60 minutes in the same room with the mechanical device producing smoke from cigarettes containing 16 mg of tar and 1.6 mg of nicotine per cigarette at the rate of approximately 100 mls of smoke every two minutes. Carbon monoxide levels were taken in the room after 30 and 60 minutes and pre-exposure and post-exposure venous blood samples were taken and changes in carboxyhemoglobin determined. For the purposes of this study, falls of 20% or more in FEV_1, FVC and PFR, and 30% in MMEFR, over baseline levels were considered significant.

The concentration of smoke achieved for each individual subject was in the same range of 20-25 parts per million of carbon monoxide, and all subjects had similar rises in carboxyhemoglobin, 0.5 ± 0.14%. Chest tightness described as asthma was produced in all six subjects and was described as an average asthmatic attack; the sensation of chest tightness commenced within 15 minutes of smoke exposure and continued for up to one hour post-challenge. These symptoms did not occur on the non-smoke inhalation study day. There were no significant changes in the pulmonary function parameters measured in any of the subjects when compared with baseline values. The largest fall in FEV_1 was 12.55% in one subject, and FEV_1 in this subject did not return to pre-challenge levels for one hour after exposure. Another subject showed a 26.8% fall in MMEFR which also lasted for one hour post-challenge.

Thus, passive exposure to cigarette smoke in these subjects produced marked symptoms described as usual asthma but not significant objective evidence of airways obstruction.

ASSESSMENT OF BREATHLESSNESS IN ASTHMA

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We report the early results of a study conducted to gain some understanding of the variability of breathlessness in asthma. Using diary cards 21 asthmatic outpatients recorded their symptoms of breathlessness, using a category scaling technique (range; 0 = no breathlessness, 10 = maximum breathlessness), and the severity of their airflow obstruction, assessed by peak expiratory flow rate (PEFR) measurement (Wright's mini-peak flow meter), twice daily (morning and evening) for two weeks.

The results showed that breathlessness increased as PEFR decreased in 17 (81%) patients, but these indices were seemingly unrelated in four others. Despite a close linear relationship in most subjects (mean $r = 0.74 \pm 0.15$ SD; $p < 0.001$) there was considerable variation in the severity of breathlessness for any particular degree of airflow obstruction (mean intercept on sensory axis = 6.3 ± 4.6 SD). However the increase in breathlessness with increasing airflow obstruction showed little variation (mean slope 0.01 ± 0.01 SD). The variability in breathlessness in asthma is likely to have many components. In this study we were able to show: (1) that for any given reduction in PEFR, patients with airflow obstruction throughout the study period (mean PEFR < 80% predicted) were less breathless than